Meconium stained amniotic fluid

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Abstract

There are few reasons why meconium is passed by the fetus *in utero*. One is a function of fetal maturity, but it may also indicate possible fetal compromise. Other reasons include the use of misoprostol, a prostaglandin (PG E1) given for induction of labour. PGs can increase the intestinal motility of the fetus and stimulate the fetus to pass meconium. Fetal infection is also associated with the passage of meconium. The incidence of meconium staining of the liquor increases from 36 to 42 weeks gestation, reaching around 20–30% at full term. It can therefore be a marker of maturation of the central nervous system and the gastrointestinal system. Passage of meconium in the preterm fetus should raise the possibility of intrauterine infection such as listeriosis. Meconium can be aspirated *in utero* or after birth. Although fetuses do not normally draw amniotic fluid into the airway, they gasp when hypoxic and therefore the coexistence of hypoxia and acidosis may precipitate meconium aspiration. Asphyxia (i.e. hypoxia and metabolic acidosis) causes added damage to the lungs and therefore further complicates the management of MAS. Aggressive tracheal toilet at delivery helps to reduce the load but will not significantly impact on cases with pre-existing *in utero* aspiration. Meconium aspiration can happen *in utero* and this is precipitated by gasping of the fetus as a result of hypoxia. The incidence of abnormal fetal heart rate patterns, increased incidence of operative deliveries for impending fetal hypoxia, and poor neonatal outcomes is greater when there is thick meconium stained liquor. Hence the presence of thick meconium needs increased vigilance with continuous electronic fetal monitoring and a lower threshold for action with CTG abnormalities.

Keywords fetal compromise; fetal hypoxia; fetal infection; meconium; meconium aspiration syndrome; prostaglandins

Introduction

Meconium is comprised of gastrointestinal secretions, bile, mucus, vernix caseosa, lanugo hair, cellular debris and amniotic fluid. It has a dark green colour, high viscosity and accumulates in the fetal intestinal tract during the 3rd trimester of pregnancy, being the first intestinal discharge released within 48 hours after birth.

Antenatal or intrapartum meconium release is referred to as meconium staining of amniotic fluid (MSAF) and it has been estimated to occur in approximately 13% of all live births with rates reported between 8 and 20%. In post-term pregnancies it is much more common as studies show rates as high as 23–52% in pregnancies at 42 weeks and 27.1% in pregnancies at 41 weeks' gestation. Preterm births are not frequently associated with meconium and it is estimated that only 5% are complicated by MSAF. Intrauterine growth restriction is also a risk factor for MSAF, as higher rates have been reported in such populations of neonates. Overall rates are on a decline in recent years, as post-term pregnancies are reduced by induction at 41 weeks and advances have been made by early intervention with an abnormal fetal heart rate.

Traditionally, three grades of meconium are described: Grade 1 meconium (light) is diluted by a large volume of amniotic fluid which is lightly stained by meconium, Grade 2 (moderate) meconium is a reasonable amount of amniotic fluid with a heavy suspension of meconium and Grade 3 meconium (thick meconium) is in little amniotic fluid suggesting the presence of meconium in scanty amounts of amniotic fluid. If no amniotic fluid is obtained at artificial rupture of membranes, one should consider the possibility of Grade 3 meconium behind the fetus and the fetal condition should be observed closely. The significance of meconium varies with presentation. With a breech presentation in the late first or second stage the passage of meconium is likely to be due to mechanical causes and therefore less sinister than a cephalic presentation, or when it occurs in an early breech labour with a high presenting part. The recent NICE guidelines suggest that the presence or absence of meconium should be documented. Significant meconium has been defined as dark green or black fluid with thick clumps of meconium or tenacious or gelatinous type of meconium. Continuous monitoring and facilities for neonatal resuscitation have been recommended.

Pathophysiology

Fetal maturity

Fetal gastrointestinal maturation is probably responsible for meconium release in post-term pregnancies as the innervation of the anal sphincter is complete by the 34th week. MSAF is rare earlier than 34 weeks' gestation. Fetal defaecation is believed to be a normal process, as studies that have assessed the diameter and function of the fetal anus sonographically over the course of pregnancy have reported at least one episode of defaecation in all fetuses and that this is independent of labour. A study by Oyelese et al., retrospectively assessed meconium staining and umbilical artery acid-base balance of 766 neonates complicated by MSAF, and concluded that there is no variation in umbilical artery pH, and that the rate of MSAF increased significantly as the gestation advances with an incidence of only 1.2% by 32 weeks.

Fetal hypoxia

Several studies suggest that hypoxia, primarily due to umbilical cord compression, promotes meconium release. An increased vagal outflow is believed to stimulate intestinal peristalsis and anal sphincter relaxation. Increased parasympathetic tone is responsible for motilin increase. A study assessing oxygen saturation at the time of meconium passage reported associated hypoxia. The type of hypoxia that induces meconium release is

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not yet clear as experimental animal studies showed variable outcomes. In a study of pregnant rats, acute hypoxia or normoxia was induced for 35 minutes followed by immediate delivery of all fetuses. Amniotic fluid bilirubin, intestinal alkaline phosphatase, plasma corticosterone releasing factor (CRF) and corticosterone (maternal and neonatal) were measured in both groups. In the acute hypoxia group all fetuses showed signs of defaecation, with significantly higher bilirubin levels, and higher alkaline phosphatase, CRF and corticosterone than the normoxia group. Induced repeated hypoxia in a group of fetal sheep, did not show association with meconium passage, but in a group that underwent chemical sympathectomy, all had thick meconium passage. This study suggests that unless there is a reduction in neural tone, there is no direct association between acute hypoxia and MSAF. Chronic hypoxia instead of acute, has also been proposed as a possible mechanism for meconium passage. In a study of 56 women in spontaneous term birth, umbilical artery cord pH, lactate, hypoxanthine and erythropoietin were measured. In neonates born with MSAF, erythropoietin was significantly higher than those born with clear amniotic fluid, while the other markers of acute hypoxia showed no significant difference between the two groups.

Risk factors

Onset of labour

Several studies suggest that the presence of labour significantly increases the incidence of MSAF. A retrospective study by Lee et al., reported a rate of 2.8% before the onset of labour and 23.1% after the onset of labour. It is also associated with prolonged or obstructed labour. Fetal plasma levels of corticotrophin release factor hormone (CRF) and cortisol are increased after 36−37 weeks and especially after the onset of labour, as a response to fetal stress. CRF is believed to be associated with colonic activity as suggested by the fetal rat studies, which explains the higher prevalence of MSAF during labour.

Prelabour rupture of membranes (PROM)-infection

In cases of PROM, the risk of chorioamnionitis is substantially increased. The prevalence of MSAF is slightly increased with clinical chorioamnionitis. This finding was confirmed by Romero et al., who showed more positive microbiological cultures with MSAF compared with clear amniotic fluid in cases of PROM (33% vs 11%). Meconium staining is therefore a marker for possible microbial infection as well as for preterm birth. In another cross-sectional study, it was confirmed that bacteria and interleukin 6 were significantly higher in MSAF cases than those with clear fluid. These results confirm the findings from previous studies that inflammatory cytokines (IL-1, IL-6) have an association with MSAF.

Intrauterine growth restriction (IUGR)

A higher prevalence of MSAF in the IUGR population has been reported by several authors. Gupta et al., reported that the prevalence in the low birth weight group is higher than in the normal birth weight group (17.3% vs 13.4% respectively). Similar results have been reported by a population based study of 9583 small for gestational age neonates. The rate of MSAF amongst SGA neonates was 16.6% which is independently associated with fetal distress.

Intrapartum management

Amnioinfusion

Instillation of 500−800 ml of warm saline into the amniotic cavity and a continuous infusion of up to 1500 ml, via a transcervical catheter, has been proposed as a method of reducing adverse outcomes secondary to MSAF. The proposed mechanisms are (i) dilution of thick meconium, reducing its inflammatory and mechanical effects on the respiratory system of the new born, and (ii) cushioning of the umbilical cord, which reduces cord compression and subsequent hypoxia. Studies conducted in various settings and populations showed variable results regarding the effectiveness of this intervention. A randomized trial conducted in Zimbabwe, in a setting with no intrapartum fetal heart rate monitoring, showed significant reduction in the number of MAS cases and perinatal deaths in the intervention group (MAS 3.1% vs 12.8%). In low resource settings, this technique may therefore offer some benefits. A subsequent multicenter randomized study compared neonatal outcomes following amnioinfusion for labours complicated by MSAF against a control group where amnioinfusion was not employed. Both groups were monitored by intrapartum fetal heart rate tracing and fetal blood sampling. In the amnioinfusion group of neonates, composite primary outcomes (MAS, perinatal death) did not vary significantly from the control group. Moderate and severe MAS was estimated at 4.4% in the intervention group and 3.1% in the control group and there was no difference in the risk of perinatal death. Rates of abnormal umbilical artery pH (pH <7.15) were also similar. Operative deliveries occurred in 13.5% of the intervention group and 12.1% of the control group. This study concludes that in a setting which provides intrapartum electronic fetal heart rate monitoring, amnioinfusion is not of benefit. Based on similar findings in other studies the American College of O&G (ACOG) recommended that if intrapartum monitoring is available, amnioinfusion should not be offered routinely, but remains an option in cases of repetitive variable decelerations, regardless of the presence of meconium.

Fetal heart rate monitoring

Since evidence show that meconium passage may indicate acute or chronic hypoxia, continuous fetal heart rate monitoring is recommended in these cases to detect possible fetal compromise. A prospective study of nearly 700 births investigated the correlation between fetal heart rate patterns, MSAF, umbilical artery pH and Apgar score. It concluded that MSAF improved the positive predictive value of abnormal FHR traces, but large pH variations remained unexplained. Another study that assessed the predictive values of intrapartum cardiotocography, reported that sensitivity and positive predictive value for fetal acidaemia are increased in the presence of meconium. Moderate and thick meconium, has been linked to abnormal FHR traces and adverse neonatal outcomes. A case control study reported a